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Editor
C. W. MACCHARLES, M.D. (MAN.)
Advisory Editor
ROSS B. MITCHELL, B.A., M.D., C.M. (MAN.), F.R.C.P.(C.)
Business Manager
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EDITORIAL

The Epidemic of Anterior Poliomyelitis

For several weeks now there has been an outbreak of Anterior Poliomyelitis in Manitoba of epidemic proportions. In view of this situation the Winnipeg Medical Society gave over their first meeting of the season on September 18th to a symposium on Anterior Poliomyelitis; the papers and the resumé of the subsequent discussion are published in the clinical section.

This meeting had the largest attendance of any of the Winnipeg Medical Society meetings for some time. Those who had special experience with the epidemic of 1928 discussed the features of the disease at that time, and the medical men who are engaged in combatting the present epidemic described their experience with the disease in the past few weeks. Most of the discussion dealt with the control of the disease during this acute stage, but A. A. Murray, F.R.C.S. (C.), raised the question of making provision of indigent cases with residual paralysis. It was understood that the Department of Health are initiating a programme for the care of these cases as the necessity arises.

F. W. Jackson, D.P.H. (Tor.), Deputy Minister of Health of Manitoba, congratulated the Executive of the Winnipeg Medical Society for arranging this excellent symposium on poliomyelitis. It remains, however, to record what everyone at the meeting must have felt, and that is the appreciation of the prompt and energetic efforts made by the medical officers of the Department of Health of Manitoba during the present epidemic. Every general practitioner who has come into daily contact with the necessity of diagnosing and treating anterior poliomyelitis must be grateful for the assistance which was given to him by the officers of this Department.

It is apparent that the opinions expressed by those who read papers are probably only tentative; they may be confirmed or modified, particularly the value of convalescent serum, when all the statistics are available at a later date.

—C. W. MacC.

Minutes of Executive Meeting

Minutes of a Meeting of the Retiring Executive and the Executive Elect of the Manitoba Medical Association, held in the Medical Arts Club Rooms on Friday, September 18th, 1936, at 6.30 p.m.

Present.

Dr. F. G. McGuinness	Dr. C. W. Wiebe
Dr. Geo. Clingan	Dr. W. E. R. Coad
Dr. F. W. Jackson	Dr. C. W. Burns
Dr. E. S. Moorhead	Dr. S. Bardal
Dr. F. A. Benner	Dr. J. F. Wood
Dr. O. C. Trainor	Dr. P. H. T. Thorlakson
Dr. S. G. Herbert	Dr. E. K. Cunningham
Dr. F. D. McKenty	Dr. W. S. Peters

Dr. R. E. Dicks

Following dinner the Chairman, Dr. F. G. McGuinness, called the meeting to order and requested that the minutes of the last Executive Meeting held on May 13th be read by the Secretary.

It was moved by Dr. F. D. McKenty, seconded by Dr. W. E. R. Coad: That these minutes, having been published in the Review, be taken as read. —Carried.

Dr. F. G. McGuinness, Retiring President, expressed appreciation to the members of the Executive for their support during the past year, and turned the business of the meeting over to the incoming President, Dr. Geo. Clingan, who replied to Dr. McGuinness' remarks.

Dr. Clingan advised that Dr. Moorhead wished to address the meeting.

Dr. Moorhead reported on certain negotiations he had with the Unemployment Relief Department re. refractions; copy of the report is on file. He also advised that, owing to the great demand for statistics of the medical relief scheme, it would be necessary, in his opinion, to employ an extra stenographer in order that the statistics might be of the greatest possible value.

Appointment of Committee on Federation.

Dr. Jackson read resolution passed at the meeting of the Canadian Medical Association Council in Victoria, and advised that Dr. Routley would be passing through Winnipeg next Friday, and it was essential that the Committee be appointed now.

Following discussion, is was moved by Dr. F. G. McGuinness, seconded by Dr. F. A. Benner: That Dr. F. D. McKenty be Chairman of Committee on Federation with power to add, and pick his own committee. —Carried.

Dr. Moorhead stated that some consideration should be given to this before October 29th, so that this could be presented to the Executive Committee of the Canadian Medical Association at its meeting by our representative on the Canadian Medical Association Executive.

Date and Place of Annual Meeting.

It was decided unanimously that the next Annual Meeting of the Association should be held in the spring, the date of the meeting to be carefully considered and brought up at the next meeting of the Executive.

Letter from Dr. C. M. Strong.

The Secretary read a communication from Dr. Strong, under date of July 11th, in connection with the right of workmen to choose their own doctor instead of being intimidated to going to Company doctors. Dr. Strong's letter was discussed and it was decided that this matter be left with the Winnipeg members of the Executive to deal with.

Dr. James McKenty's Report on "The Relation of the Profession with Hospitals.

The Secretary read a letter from Dr. A. W. S. Hay, Secretary of the Winnipeg Medical Society, under date of March 23rd, attaching Dr. McKenty's paper on the above subject.

It was stated that this was previously held in abeyance, as it was thought the time was not suitable for action on it. Since this, however, Dr. McKenty has had this article published in full in the "Canadian Doctor." Following discussion, it was decided that a letter should be written to the Winnipeg Medical Society, asking for details as to the disposition of this matter.

Letter from Dr. J. W. Cairns.

The Secretary read communication from Dr. Cairns of Pipestone under date of September 2nd, advising that the Federal Government had taken over the whole relief in that district and the set-up did not include medical services. Dr. Jackson explained the situation and advised that information has been gathered as to what the cost would likely be, as this affects eight municipalities. It was suggested that this matter be brought to the attention of Dr. T. C. Routley on his way through Winnipeg this week, and ask him to keep in close touch with the matter at Ottawa and make sure that provision is made for medical attendance.

It was moved by Dr. J. F. Wood, seconded by Dr. S. G. Herbert: That this be left to the Secretary to take care of and reported back to the next meeting of the Executive. —Carried.

Letter from Medical Council of Canada Re. Specialists.

The Secretary read a communication from The Medical Council of Canada and resolution passed by that body on the control of Specialists in Canada. As the members of the Executive were not familiar with this matter, it was suggested that copies of the report of this Committee and information on the subject be prepared and forwarded to each member of the Executive, and that the matter be brought up at a future meeting.

Letter from Department of Health and Public Welfare: Re. Training Schools for Nurses.

The Secretary read a communication dated September 17th from the Department of Health and Public Welfare, regarding training schools for nurses, stating that the University of Manitoba requested that they be free from any responsibility of designating what hospitals should be allowed to operate training schools. The Registered Nurses Association were making a survey and the Department of Health and Public Welfare wished the opinion of the medical profession. Following discussion, it was decided that a questionnaire should be prepared and brought for consideration at next executive meeting.

There being a meeting of the Winnipeg Medical Society held this evening at 8.30 p.m., the meeting adjourned and a further meeting of this Executive to be called in one month's time.

University of Manitoba

FACULTY OF MEDICINE

POST-GRADUATE COURSE 1936-37: CLINICAL ENDOCRINOLOGY

Nov. 18th—Professor A. T. Cameron—Thyroid (General).

Nov. 25th—H. D. Kitchen, M.D. (Man.)—Non-myxoedematous sub-thyroid states and medical treatment of hyperthyroid states.

Nov. 25th—G. S. Fahrni, F.R.C.S. (C.)—Surgical treatment of hyperthyroidism.

Dec. 2nd—Professor A. T. Cameron—Parathyroid, Thymus, Pineal (General).

Dec. 9th—Bruce Chown, B.A., M.D. (Man.)—Recognition and medical treatment of sub- and hyperparathyroid states.

Dec. 9th—A. C. Abbott, F.R.C.S. (Edin.)—Treatment of hyperparathyroid states including those associated with kidney lesions.

Jan. 13th—Professor A. T. Cameron—Pancreas (General).

Jan. 20th—Professor C. R. Gilmour—Hypoglycaemia.

Jan. 20th—A. Hollenberg, B.Sc., M.D. (Man.)—Treatment of Diabetes.

Jan. 27th—Professor A. T. Cameron—Reproduction (General).

Feb. 3rd—Time reserved for guest speaker.

Feb. 3rd—J. D. McQueen, F.R.C.S. (C.)—Treatment of disturbances of the menstrual cycle.

Feb. 10th—Professor A. T. Cameron—The Adrenal Glands (General).

Feb. 17th—J. D. Adamson, M.R.C.P. (Edin.)—Addison's Disease.

Feb. 17th—Professor J. A. Gunn—Paroxysmal hypertension.

Feb. 24th—Professor A. T. Cameron—The pituitary gland (General).

Mar. 3rd—Lennox G. Bell, M.R.C.P. (Lond.)—Cushing's syndrome, acromegaly, dwarfism.

Mar. 3rd—O. S. Waugh, F.R.C.S. (C.)—Surgical treatment of pituitary conditions.

Mar. 3rd—John Hillsman, Ch.M. (Man.)—The pituitary and water regulations.

Mar. 10th—Professor A. T. Cameron—Anti-hormones and pituitary inter-relationships.

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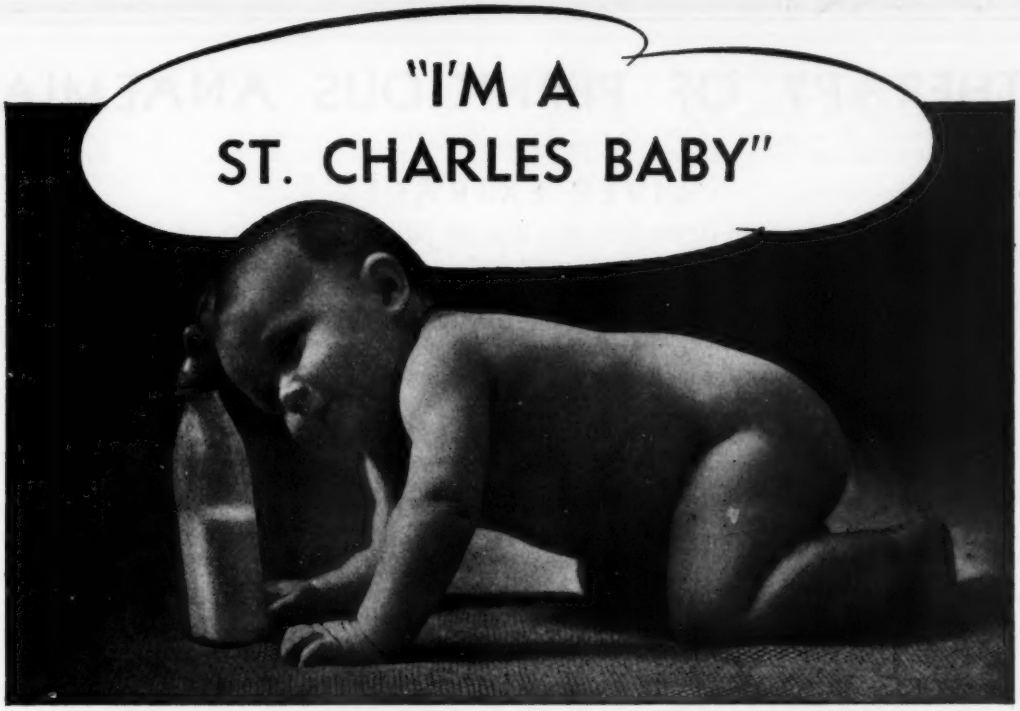

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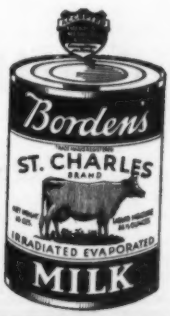

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Liver Extract of two strengths is supplied by the Connaught Laboratories. For moderate cases, satisfactory results are obtained from a solution containing, in each cc., the active substance derived from ten grams of liver. As regards patients suffering from the spinal cord lesions of Pernicious Anaemia, recent studies have shown that they do best when given prolonged rest and much larger amounts of liver or liver extract than are usually required to relieve the anaemia of the disease. A solution of liver extract which clinical studies have shown to be particularly effective in the treatment of such patients is one that has been prepared in the Connaught Laboratories to contain, in each cc., extract from twenty grams of liver. This solution enables adequate amounts of active substance to be administered in relatively small volumes.

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NEWS ITEMS

The following is an article written on "Intestinal Intoxication—its Prevention," by Samuel Karelitz. This appeared in the August, 1936, copy of "Preventive Medicine."

"In recent years it has become apparent that the severe symptoms which make up the picture of intestinal intoxication are the result of a disturbance in the chemical equilibrium in the body brought about by the loss of water, salts and organic substances, primarily by way of the gastrointestinal tract, and that the duration and severity of the diarrhea determine the appearance of the clinical picture of intoxication rather than the underlying cause of the initial gastrointestinal upset. Expressed differently, any condition which leads to diarrhea in infancy if permitted to continue will ultimately bring about the state of anhydremia (desiccation or severe water loss) with its resulting blood concentration, acidosis, toxemia and circulatory collapse.

The above facts being the generally accepted views concerning intestinal intoxication, a paper dealing with the preventive aspects of the symptom complex should, therefore, deal with the causes, the prophylaxis, and the treatment of the ordinary gastrointestinal upsets of infancy, especially diarrhea.

CAUSES OF DIARRHEA

The classification of the causes of gastrointestinal disturbances in infancy made by Czerny serves well in the discussion of the subject. He divided the causes of gastrointestinal disturbances of infancy into three groups; those associated with infection, alimentation, and those associated with constitutional peculiarities.

Under the subtitle infection he referred to the harmful effect of enteral infection, such as dysentery; the parenteral infections such as acute upper respiratory infections, otitis media, and pneumonia, and to the injurious substances introduced with food, which had been caused by the bacterial action upon it, or by the harmful products of fermentation and putrefaction brought about by the action of bacteria upon foods within the intestinal tract.

Under alimentary causes he referred to overfeeding, underfeeding, or partial starvation leading to malnutrition, or the feeding of poorly balanced foods, such as those unusually high in the fat or carbohydrate content, or the giving of food difficult for an infant to digest.

Under the title of constitutional predispositions to gastrointestinal disturbances he referred to the cases of inherent intolerence for carbohydrates or fats in quantities usually well tolerated by normal infants, to the hypertonic and pylorospastic infants, and to those now known as allergic.

The causes mentioned, and others which fall under Czerny's classification include most of the causes of gastrointestinal upsets in infancy. I have seen cases of diarrhea illustrating each of these etiological factors which have gone on to the development of intestinal intoxication.

Other causes of diarrhea which have resulted in intestinal intoxication which have come to my attention are the following: a single dose of castor oil, and in another instance a large dose of milk of magnesia given for constipation, brought about diarrhea; sudden weaning of a baby, especially in the summer, even though pasteurized milk was used; an ounce of orange juice given for the first time—the addition of a fairly large quantity of a new food, such as vegetable; general anaesthetic; mechanical injury to the intestine, such as operation, and quite commonly summer heat, especially when associated with great humidity, or with an abrupt drop in barometric pressure. It is often difficult to determine the exact etiology of a case of diarrhea, for several factors may interplay in bringing about the disturbance.

MECHANISM OF DIARRHEA

In brief, except for the direct action of enteral infection upon the intestinal tract, and the increased activity brought about by nervous stimulation as in the hypertonic or allergic child, diarrhea is produced by either bacterial irritation or by mechanical irritants to the gastrointestinal tract.

Infants in general, and artificially fed infants especially, are more likely to develop diarrhea than are older individuals. This may be due to a number of factors. The gastrointestinal secretions of infants contain less hydrochloric acid and less of the proteolytic enzymes than those of adults. The gastric juice of infants is well able to digest the small fine curds of breast milk, but is not always adequate for complete digestion of the coarse, tough cow's milk curd. The acidity of the infant's gastric juice is ordinarily adequate to prevent the growth of bacteria in the stomach as well as in the upper part of the intestinal tract. Under abnormal conditions, such as parenteral infection, long standing malnutrition (even in breast-fed infants), or abnormal climatic conditions (high temperature and humidity, and sudden drops in barometric pressure) there is a diminution in the gastric acidity. Associated with this diminished gastric acidity the colon bacillus, which is normally found as a harmless inhabitant of the lower bowel, is found in fairly large numbers in the duodenum, and even in the stomach, and by its action on foods and perhaps directly on the intestinal wall, is believed to bring about diarrhea.

Direct mechanical irritation due to foods which the particular infant is not able to digest properly, for example, large, tough milk curds, highly concentrated sugar solutions, the lower fatty acids, coarse vegetable particles, and toxic products of fermentation and putrefaction, may bring about diarrhea.

PROPHYLAXIS OF DIARRHEA

Despite the fact that diarrhea in most instances is a preventable condition, diarrheal conditions in infancy are still important contributing factors, directly or indirectly, to infant mortality. Probably the best prophylactic measure is breast feeding, since human milk is digestible in nearly all infants. By breast feeding the introduction of harmful bacteria is avoided and malnutrition, one of the greatest predisposing factors to diarrhea, and therefore to intestinal intoxication, is less likely.

Pasteurization and boiling of milk are the most important of the prophylactic measures in the prevention of diarrhea in artificially fed infants. Boiling is especially helpful, for in addition to the destruction of bacteria, the milk is rendered more digestible. Boiling of pasteurized milk for one or two minutes is an important safeguard against possible contamination of the milk after it has been bottled.

Evaporated milk can serve as an excellent substitute for pasteurized milk, and has the added advantages of being less costly for those who are unable to afford grade A milk, of being more digestible for premature infants, and less disturbing in many cases of milk allergy. Because of the greater digestibility of evaporated milk it can be used in greater concentration for premature and feeble infants, thus avoiding the possibility of malnutrition. Powdered milks are useful in that they are more digestible and are easily portable during travel.

An adequate daily water intake must be guaranteed. The infant requires a minimum daily fluid intake of approximately 10 per cent of its body weight. Fifteen per cent is optimum under ordinary conditions, and 20 per cent is maximum, unless the weather is unusually hot and conducive to very great water loss by sweating. The infant usually gets its essential fluid requirement in its food. If, however, there is exessive water loss, either as a result of increased body temperature, or as a result of an increased external temperature, extra water must be offered between feedings to avoid dehydration. In like manner adjustment of clothing and room temperature and air circulation is necessary to reduce excessive water loss in the summer.

The use of correct milk modifiers has also been an important factor in the reduction of diarrhea. Dilution of milk with water aids in reducing the size of the milk curd but unless done intelligently is associated with the danger of underfeeding. Acidification of milk by the use of lactic acid is helpful in that it reduces the size of the curd and by increasing the gastric acidity aids in the prevention of bacterial proliferation in the stomach and in the duodenum. In some parts of the United States a formula of acidified evaporated milk, water and karo syrup has become very popular and the results obtained through its use have been quite satisfactory.

The choice of a carbohydrate modifier is under ordinary conditions important only in that it should not exceed 10 per cent of the total volume of the formula. A combination of a simple sugar and a starch totalling 6-7 per cent of the total volume of the formula is most often successful. If the carbohydrate used in the formula seems to be inadequate a change to a different one, preferably one like dextrimaltose, usually suffices.

Finally the factor which most often initiates diarrhea, namely, parenteral infection, must be dealt with promptly and adequately. For example, in the case of acute otitis media it may be wiser to incise the ear drum in the presence of high temperature, severe earache, restlessness, anorexia and vomiting even though the usual indications for paracentesis have not fully developed.

TREATMENT OF DIARRHEA

Since the development of intestinal intoxication depends on the outcome of the treatment of a case of diarrhea, and since diarrhea will occur inevitably despite all efforts to prevent it, this phase of the subject is extremely important. The reduction in the number of cases of intestinal intoxication in the practice of most physicians would indicate that the treatment of simple diarrhea is fairly well and generally understood. Intestinal intoxication still does occur and is associated with a high mortality.

Certain principles of treatment apply to all forms of diarrhea. These are the early recognition and prompt treatment of parenteral infections; rest of the gastrointestinal tract; restoration and maintenance of a fluid and mineral balance; the giving of food adapted to the limited digestive capacity.

Diarrhea in the breast-fed is usually mild, and is treated by merely reducing the period and frequency of nursing until remission takes place. In the partially weaned baby it is best to eliminate the supplemental feedings entirely, and only gradually replace them when the diarrhea is improved.

In artificially fed infants the treatment of diarrhea is more complicated. In the very mild case it may be adequate to eliminate fruits, vegetables, and other accessories such as cod liver oil until improvement is attained. In those more severe, treatment must be more drastic. First the cause should be determined if possible, and if infection is present it should be promptly and specifically treated as is indicated.

In any case of diarrhea the rate of evacuation of the intestinal content is increased and in consequence digestion and absorption are impaired. Under these conditions, giving the usual diet can only do harm by increasing the already existing irritation of the gastrointestinal tract. It is necessary therefore to diminish food in the very mild cases and in the more severe to withhold food entirely, (for periods varying with the intensity of the condition) from one to two feedings in the relatively mild, to 24 hours or longer in the more severe cases. Irritation of the rectum by the insertion of thermometers or rectal tubes should be avoided. Cathartics should be avoided except for those relatively infrequent cases of diarrhea seen early and known to be due to a food irritant, in which cases a single small dose of castor oil or an enema may be helpful. Although this paper deals with a condition occurring in infancy, the rare case of appendicitis should be kept in mind in giving any cathartic.

During the period of starvation fluid balance should be maintained. Water as such or 5 per cent barley or rice flour solutions, or weakened tea, plain or with the addition of 5 per cent added glucose may be administered by mouth. If vomiting or severe abdominal distention are present the fluid requirement may be satisfied by the subcutaneous and intravenous injection of either Ringer's or Hartmann's or physiologic sodium chloride solution. For intravenous injection 5 to 10 per cent added glucose is helpful. If it is necessary to maintain the intravenous injection over a long period a rate of approximately 130-150 c.c. per kilogram of body weight per 24 hours is usually adequate.

When vomiting has ceased and diarrhea has improved feeding is resumed and the quantities and type of food should depend on the severity of the condition. Fat is least tolerated under these conditions. The choice of formula should therefore be made with that in mind. Sour milk, such as buttermilk or protein milk are generally useful, to be replaced by sweet milk mixtures when the diarrhea improves. It is perfectly possible to resume feeding with sweet milk mixtures provided the quantity offered is very small and the daily increases are likewise small. Thus, in the severe cases 10 c.c. of a 2/3 milk, 1/3 water plus 5 per cent added sugar every 2 hours and increased by an equal amount daily has been found to be generally successful. In relatively few instances was protein milk or buttermilk found to be better than sweet milk mixtures when administered as directed above.

In the course of treatment of a case of diarrhea if symptoms such as loss of tissue turgor, fever, loss of lustre of the eyes and continued or increased diarrhea occurs, intestinal intoxication is present. In that case starvation for at least 36 hours, continuous intravenous injections of the fluids mentioned is instituted immediately and maintained until detoxification is complete and until the child is able to take an adequate amount of fluid by mouth. Blood transfusion is regularly given soon after the intravenous drip has been started. We have found it wise not to inject blood if the hemoglobin content is greater that 80 per cent, or until it falls below that level. Feeding in the manner previously referred to for severe cases of diarrhea, is started only after detoxification is complete, abdominal distention, if present, is relieved, vomiting has ceased, and the diarrhea improved.

I have attempted to show that it is possible in one's general practice to avoid to a large extent the occurrence of diarrhea, to treat it adequately when it occurs, and how to cope with the very severe cases which resist the ordinary treatment and become toxic.

COMMUNICABLE DISEASES REPORTED
Urban and Rural - August, 1936.

Occurring in the Municipalities of:

Scarlet Fever: Total 121—Winnipeg 84, St. Vital 8, Argyle 4, Winnipeg Beach 4, Ste. Anne 3, Dauphin Town 2, Gilbert Plains Rural 2, Portage Rural 2, Brandon 1, Fort Garry 1, Hanover 1, La Broquerie 1, Oakland 1, Portage City 1, St. Andrews 1, St. Boniface 1, St. James 1, St. Paul East 1, Tache 1, Transcona 1.

Measles: Total 83—Virden 17, Unorganized 14, Wallace 11, Winnipeg 10, Eriksdale 6, Thompson 4, Oak Lake 3, Bifrost 2, Brokenhead 2, Minto 2, Archie 1, Elkhorn 1, Emerson 1, Flin Flon 1, Harrison 1, Pipestone 1, Portage City 1, Sifton 1, Springfield 1 (Late Reported: May, St. Clements 1, June, Portage Rural 1, St. Andrews 1).

Anterior Poliomyelitis: Total 62—Morton 17, Boissevain 12, Whitewater 6, Stanley 4, Winnipeg 3, Gretna 2, Morden 2, Wawanesa 2, Brandon 1, Cartier 1, Kildonan West 1, Killarney 1, Louise 1, Manitou 1, Norfolk North 1, Pembina 1, Portage Rural 1, Riverside 1, Rockwood 1, Selkirk 1, Turtle Mountain 1 (Late Reported: July, Montcalm 1).

Tuberculosis: Total 43—Winnipeg 13, Unorganized 3, Brandon 2, Dauphin Rural 2, Armstrong 1, Birtle Rural 1, Blanshard 1, Dauphin Town 1, Eriksdale 1, Ethelbert 1, Flin Flon 1, Gilbert Plains Rural 1, Grandview Town 1, Hanover 1, Minitonas 1, Portage Rural 1, Rhineland 1, Rockwood 1, Rossburn Town 1, Selkirk 1, Siglunes 1, Springfield 1, Stonewall 1, St. Boniface 1, St. James 1, Tache 1, The Pas 1.

Whooping Cough: Total 28—Winnipeg 13, St. Paul East 7, Eriksdale 3, Franklin 3, Killarney 1, Unorganized 1.

Chicken Pox: Total 22—Winnipeg 15, St. Clements 3, St. Boniface 2, Blanshard 1, Victoria Beach 1.

Diphtheria: Total 12—Winnipeg 6, Rivers 2, Winnipeg Beach 2, Portage City 1, Whitewater 1.

Mumps: Total 7—Winnipeg 4, Springfield 1, St. James 1 (Late Reported: June, Morden 1).

Typhoid Fever: Total 6—Argyle 2, Hanover 2, Portage City 1, Winnipeg 1.

German Measles: Total 5—Flin Flon 1, Roland 1, St. Andrews 1, The Pas 1 (Late Reported: June, Clanwilliam 1).

Erysipelas: Total 5—St. Clements 2, Unorganized 1, Woodlands 1, Winnipeg 1.

Influenza: Total 5—(Late Reported: May, Unorganized 1, June, Morden 1, Portage Rural 1, Unorganized 2).

Lethargic Encephalitis: Total 1—(Late Reported: May, Ethelbert 1).

Puerperal Fever: Total 1—Harrison 1.

Trachoma: Total 1—Winnipeg 1.

Amoebic Dysentery: Total 1—(Late Reported: June, Arthur 1).

Undulant Fever: Total 1—Winnipeg 1.

Diphtheria Carriers: Total 1—Winnipeg 1.

Venereal Disease: Total 133—Gonorrhoea 95, Syphilis 38.

DEATHS FROM ALL CAUSES IN MANITOBA
For the Month of July, 1936.

URBAN—Cancer 46, Pneumonia 13, Tuberculosis 5, Syphilis 2, Influenza 1, Puerperal Septicaemia 1, Typhoid Fever 1, all other causes 203, Stillbirths 9. Total 281.

RURAL—Pneumonia 19, Cancer 16, Tuberculosis 13, Measles 3, Infantile Paralysis 2, Influenza 2, Lethargic Encephalitis 1, Puerperal Septicaemia 1, Whooping Cough 1, Erysipelas 1, Syphilis 1, all others under 1 year 6, all other causes 168, Stillbirths 8. Total 242.

INDIAN—Tuberculosis 10, Measles 2, Pneumonia 1, all others under 1 year 2, all other causes 8. Total 23.

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"Endocrines and Mental Disorders"—by Sir W. Langdon-Brown, M.A., M.D., F.R.C.P., St. Bartholomew's Hospital.

"Classification and Etiology of the Neuroses"—by Hugh Crichton Miller, M.A., M.D., M.R.C.P.

"Early Symptoms of Mental Disorders"—by R. D. Gillespie, M.D., F.R.C.P., Guy's Hospital.

"The Physical Factor in Mental Disorder"—by J. G. Porter Phillips, M.D., F.R.C.P., London.

"Depression"—by W. R. Reynell, M.A., M.D., F.R.C.P.

"Dementia Praecox"—by Daniel F. Rambant, M.A., M.D.

"Drug Addiction and Alcoholism"—by Sir James Purves-Stewart, C.M.G.C., M.D., F.R.C.P.

"Sexual Perversions"—by J. R. Rees, M.D., London.

"The Practitioner"—August, 1936.

This issue contains a symposium on Venereal Disease.

"Some late Effects of Venereal Disease"—by L. W. Harrison, D.S.O., M.B., Ch.B., F.R.C.P. (E.).

"The Diagnosis and Treatment of Acute or Early Syphilis"—by E. T. Burke, D.S.O., M.B., London.

"The Diagnosis and Treatment of the Local Complications of Gonorrhoea in the Male"—by V. E. Lloyd, M.C., M.B., B.S., Guy's Hospital.

"Gonorrhoea in Women"—by Margaret Rorke, M.B., Ch.B., M.C.O.G.

"Virus Diseases of the External Genitals and Chancroid"—by Robert Lees, M.B., Ch.B., F.R.C.P. (E.), Edinburgh.

"Venereal Disease in Children"—by F. R. Curtis, M.B., Ch.B., M.Sc.

"Cancer of the Rectum"—by Sir Charles Gordon Watson, K.B. (E.), C.M.G., F.R.C.S.

"The Practitioner"—September, 1936.

This number contains a symposium on "Industrial Medicine" comprised of the following articles:

"The Prevention of Disease in Industry"—by John C. Bridge, F.R.C.S. (E.), H.M. Senior Medical Inspector of Factories.

"Physical Standards in Industrial Health"—by Sir David Munro, K.C.B., M.B., F.R.C.S. (E.).

"Industrial Absenteeism"—by N. Howard Mummery, M.R.C.S., L.R.C.P.

"Industrial Poisons"—by Donald Hunter, M.D., F.R.C.P., London Hospital.

"Industrial Diseases of the Lungs"—by W. E. Cooke, M.D., F.R.C.P., D.P.H.

"Psychological Disorders in Industry"—by Millais Culpin, M.D., F.R.C.S.

"Some Practical Aspects of Industrial Hygiene"—by Donald Stewart, M.D., M.R.C.P.

"The Prevention and Cure of Occupational Dermatitis"—by P. B. Mumford, M.D., F.R.C.P., Manchester.

"The Canadian Medical Association Journal"—September, 1936.

"Clinical Experiences with Protamine-Zinc-Insulin and Other Mixtures of Zinc and Insulin Diabetes Mellitus"—by I. M. Rabinovitch, J. S. Foster, A. F. Fowler and A. C. Corcoran.

"The Results of Sympathectomy in Children"—by John L. McDonald, M.B., University of Toronto, Toronto.

"Rheumatic Infection in Childhood: Observations on the Sedimentation Rate and the Schilling Count"—by R. R. Struthers, M.D., and H. L. Bacal, M.D., Montreal.

"The Surgeon's Responsibility in the Treatment of Duodenal Ulcer"—by Roscoe R. Graham, M.B., F.R.C.S. (C.).

"Migraine"—by David Slight, M.B., Ch.B., D.P.M., F.R.C.P. (C.).

"The Prognosis of Coronary Thrombosis"—by G. F. Strong, M.D., Vancouver.

"A Further Report on Obstetric Analgesia and Amnesia"—by Lieghton C. Conn, F.R.C.S. (C.), and John Ross Vant, M.C.O.G., Edmonton.

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"Silicosis and its Incidence in British Columbia"—by C. H. Vrooman, M.D., C.M., F.R.C.P. (C.), Vancouver.

"The American Journal of Surgery"—September, 1936.

This issue contains a number of articles forming a symposium on Gynaecology among which number are the following excellent articles:

"Amenorrhoea; Menorrhagia; Metrorrhagia; Delayed Menopause"—by H. S. Crossen, M.D, F.A.C.S. and Robert J. Crossen, St. Louis, Mo.

"Endometrial Cycle and Mechanism of Normal Menstruation"—by Somers H. Sturgis, M.D., and Joe V. Mey's M.D., Boston, Mass.

"Dysmenorrhoea"—by Albert Mathiue, M.D., F.A.C.S., Portland, Oregon.

"Sterility"—by Sophia J. Kleegman, M.D., F.A.C.S., New York.

"Operative Treatment of Sterility"—by Francis W. Sovak, M.D., F.A.C.S., New York.

"Pelvic Endometriosis and Its Treatment"—by Emil Novak, M.D., F.A.C.S., Baltimore.

"Vaginal Hysterectomy: Clamp Method for Uterine Prolapse"—by J. W. Kennedy, M.D., F.A.C.S., Philadelphia.

"Le Fort Operation for Uterine Prolapse"—by Fred L. Adair, M.D., F.A.C.S. and Laura Da Sef, M.D., Chicago.

"Treatment of Prolapse of Uterus by the Manchester-Fothergill Operation"—by Charles A. Gordon, M.D., F.A.C.S., Brooklyn, N.Y.

"Cancer of Corpus Uteri"—by William P. Healy, M.D., F.A.C.S., New York, N.Y.

"Fibromyoma Uteri"—by J. P. Greenhill, M.D., F.A.C.S., Chicago.

"Salpingitis"—by J. Randolph Gepfert, M.D., New York.

"Blood Transfusion in Gynecology"—by R. E. Stetson, M.D., New York.

"The Clinical Journal"—September, 1936.

"The Modern View of Nephritis and its Treatment"—by F. A. Roper, M.A., M.D., M.R.C.P.

"Medical Treatment of Urinary Infections"—by Reginald Ellis, M.D., M.R.C.P.

"Manipulative Surgery"—by T. McW. Millar, M.B., F.R.C.S. (E.).

"Tests for Drunkenness"—by T. H. Blench, M.D., Police Surgeon, Manchester.

"Canadian Public Health Journal"—August, 1936.

"A Recent Outbreak of Haemorrhagic Smallpox in British Columbia"—by J. W. McIntosh, B.A., M.B., D.P.H., Vancouver.

"Progress in Public Health in Canada"—by C. G. Power, B.A., Minister of Pensions and National Health, Canada.

"The Clinical Journal"—August, 1936.

"Haemorrhage in Early Pregnancy"—by A. M. Claye, M.D., F.R.C.S., Leeds.

"The Diagnosis of Some Febrile Conditions"—by G. Lovell Gulland, C.M.G., M.D., F.R.C.P. (Ed.), Edinburgh.

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OBITUARY

DR. JON STEFANSSON

Dr. Jon Stefansson died in the Winnipeg General Hospital on September 29th of pneumonia. He had been present at a celebration at Gimli on September 19th when Lord Tweedsmuir visited the pioneer Icelandic settlement, but was stricken down a day or two later.

His name was internationally known for his operation for the relief of glaucoma. He was born in Iceland, 1878, and at the age of ten came to Manitoba with his father, who settled on a farm at Cypress River. He was educated at Wesley College and the Manitoba Medical College. After an apprenticeship with the late Dr. S. W. Prowse, he spent two years in study in London, Glasgow, Vienna and Berlin, narrowly escaping internment at the outbreak of the Great War. Returning to Winnipeg, he carried on practice in diseases of the eye, ear, nose and throat.

He took a keen interest in the welfare of his Icelandic countrymen, read widely, and delighted in music. His wife, Joanna Philipowska, Russian opera singer, predeceased him, but he is survived by a son and daughter.

DR. ROBERT STIRTON THORNTON

Dr. Robert Stirton Thornton, aged seventy-three, died at Vancouver on September 18th. Although the last five years of his life were spent on the Pacific coast, his active life was lived in Manitoba, and on account of his position in the legislature and in the Masonic craft, he was almost as well known in Winnipeg as in Deloraine, where he practised from 1887-1931. He was born at Edinburgh in 1863, was educated at Herriot's College and the University of Edinburgh, and started practice shortly after graduation at Deloraine at a time when settlers were crowding into southern Manitoba. He was Chairman of the School Board at Deloraine for some years and was a prime mover in establishing the park there, which is so well known to visitors. In 1911 he was elected to the Manitoba Legislature, was defeated in 1914, but was again returned in 1918 and served as Minister of Education in the Norris Government. In this capacity he did much to unify education and to stamp out multilingualism. He planned to make the school a community centre. In 1914 he was elected President of the Medical Council of Canada, succeeding Sir Thomas Roddick. In his presidency a Dominion-wide license to practice was obtained. Queen's University honored him with the LL.D. in 1914.

The death of his wife, following a motor accident five years ago in which he, himself, was severely injured, affected him greatly.

A man of kindly and congenial disposition, a skilful physician and a statesman of clear vision, Dr. Thornton set his mark on the Canadian West.

DR. WILFRED LORNE ATKINSON

Dr. Wilfred Lorne Atkinson died at his home in Selkirk, Manitoba, on September 15 at the age of fifty. He was born at Clearwater, Manitoba, and graduated in medicine from the Manitoba University in 1911. He practised for nearly twenty-five years in Selkirk and took a leading part in the life of the community, especially in the formation of the Selkirk Golf Club. After the war he served for a time on the staff of the Tuxedo Hospital. A heart condition crippled him during the latter years of his life and to seek relief he took a long sea voyage about two years ago. He is survived by his widow.

DR. GASPARD L. MARSOLAIS

Dr. Gaspard L. Marsolais, St. Eustache, Manitoba, died at St. Boniface Hospital on September 14th. He was born at Joliet, Quebec, in 1873, and came to Manitoba as a youth of nineteen. Graduating in medicine he returned to St. Eustache where he became medical health officer for the municipality. A widow and son survive.

NOTICE

A special program of lectures and demonstrations in medicine and surgery will be held under the direction of The Mayo Foundation from November 9 to 13, inclusive. Mornings will be devoted to surgical and medical clinics. In the afternoons and evenings symposiums will be conducted on neurology, gynecology, diseases of the ear, nose and throat, laboratory procedures, emergency treatment, pediatrics, and renal diseases. In addition, a clinico-pathologic conference will be held. While this program is arranged primarily for the Fellows of the Foundation, visiting physicians are invited to attend.

NOTICE

The Municipal Hospitals of Winnipeg recently purchased a Drinker Respirator for use in various forms of respiratory failure, particularly that associated with Infantile Paralysis. It has also been used for post-operative respiratory failure and the use might be indicated in various other clinical conditions such as various forms of poisoning, etc.

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Dr. A. B. Alexander, Medical Superintendent of the Municipal Hospitals, would arrange a demonstration for any group of medical men who might be interested.

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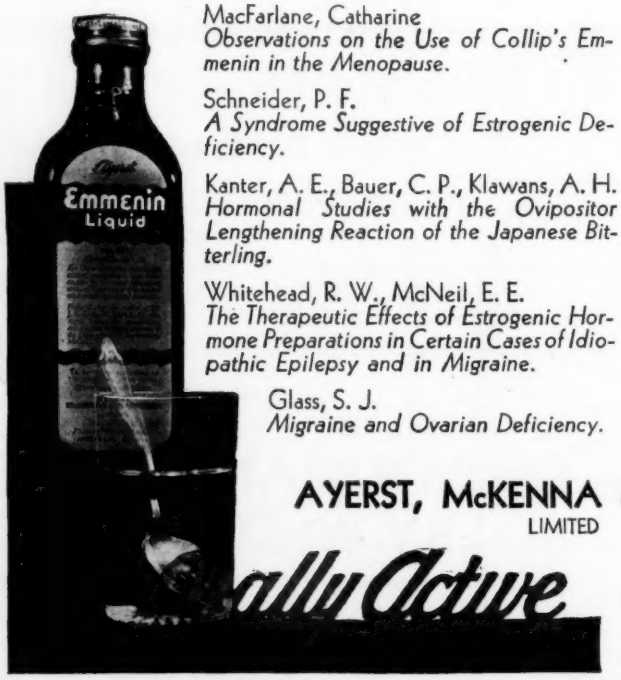

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Clinical Section

*A Clinical Study of Twenty-Five Cases of Diabetes Mellitus in Childhood

By

HARRY MEDOVY, B.A., M.D. (Man.),
Demonstrator in Medicine, University of Manitoba, Assistant Physician Winnipeg Children's Hospital and Winnipeg General Hospital

INTRODUCTION

Diabetes of childhood is practically a new disease. Before 1922 a diabetic child almost never lived more than two years after the diagnosis was made. The briefest possible mention of this condition in childhood was made in text-books and all the references simply stressed the complete hopelessness of attempting to treat the disease. With the discovery of insulin in 1922, it became possible for the diabetic child to live. It is hard to realize that a disease which fourteen years ago had a mortality rate of 100%, now has a rate of only 1% per annum (these figures from Joslin). As the diabetic children of the post-insulin era continued to live, they wrote into the text-books the story of a new disease. New problems arose — heredity, growth, adolescence, premature arterio-sclerosis, enlargement of the liver and so on. It has been my good fortune to have been able to study 25 cases of diabetes mellitus in children in the past six years and this paper is simply a recital of clinical experiences and clinical impressions arising out of this study.

In 1930, at the suggestion of Dr. Gordon Chown, a diabetic clinic was established at the Children's Hospital. The original reason for the establishment of a diabetic clinic at the Children's Hospital under a single head was for the purpose of co-ordinating and standardizing treatment. It was felt that the mortality rate was too high, that cases were being admitted and re-admitted in coma too frequently, and that there was lack of proper supervision in the home. It was felt that the children coming in in coma were treated reasonably well but that on their recovery from coma they were being given inadequate diets—diets that were often distasteful to them, and that there was an almost complete lack of knowledge at home as to how the child should be looked after. The weakest part of the link seemed to be at the time the diabetic child was discharged from hospital. For several weeks the child had been kept in the hospital, his insulin administered, his diets given to him and very accurate and voluminous charts and histories kept. Then suddenly he was sent home—in many cases with no one at home who understood the disease adequately or knew how to deal with the various contingencies as they arose. The result was that every break in diet, every overdosage with insulin, every acute infection meant a hospital visit and a stay of several weeks. The diabetic child became a "hospital child." Schooling was inadequate. Exercise was conspicuous by its absence.

The set-up for the clinic consisted of a co-ordinating group to which all public diabetic children were referred immediately upon diagnosis or admission. This consisted of:

1. *The hospital dietitian,* whose duty it was to prepare and plan the child's diet, and to teach the mother the weighing of foods and the essentials of the diet.

2. *The interne staff,* who carried out the active treatment of the cases in hospital under supervision, and who were responsible for instructing the mother or the child how to examine urine for sugar.

3. *The nursing staff,* who were responsible for accurate records and who taught the mother or the child the measuring and administration of insulin.

4. *The pathologist,* Dr. Bruce Chown, who carried out all necessary laboratory procedures.

5. *The nutritionist,* Miss Woodman, whose responsibility it was to see that no child was discharged from the hospital until someone at home was fully instructed in the care of the child. Then her job became one of follow-up to see that everything was made as easy as possible for the mother until she had developed the confidence so necessary to the proper management of the child.

6. Lastly, *The physician in charge,* who served the purpose of taking the credit for any good results that might ensue.

All this sounds very complicated and difficult and it may, perhaps, clarify matters if a typical case were followed through, from its first admission in coma. The parents have just recovered from the shock of seeing their child practically at death's door, and the awful realization that their child has an incurable disease is just dawning on them. While the patient's diet and insulin are being established, advantage is taken of the opportunity to educate the child if he is over eight years, and the mother, if he is under eight years. In children under eight years of age, the mother is asked to come down to the hospital each morning and the fundamentals of the disease are explained to her. She is told that her child will get along well and will not be denied anything but that her co-operation is essential to the successful carrying on of the case. She is told that although everything may seem discouraging and she has a great deal to learn that we will help her and see to it that everything goes well. She is taught to do a urine test for sugar and learns to appreciate the difference between a red, yellow, green and blue Benedict test. She is taken to the diet kitchen and taught how to weigh foods, how to substitute

* Presented at the Winnipeg Medical Society on October 17, 1936.

one food for another; she is asked to note down the foods commonly used in her own home and it is explained to her that her child's diet will differ very little from the diet of the rest of the family. His food will be weighed—their food will not. If the family has soup, roast beef, peas and tomatoes, pudding and milk, so will the child. Scales are bought and she is shown how to use her own scale.

Then the interne and the nursing staff take charge and explain the administration of insulin to her. She is shown how to sterilize the needle and syringe, how to measure insulin and, finally, is allowed to inject it herself under supervision. The importance of never omitting a dose of insulin is stressed. The signs and treatment of hypoglycaemia and of acute infections are taught. She is quizzed on all these points by myself, until there is complete assurance on both sides that the child is going home in competent hands.

If the child is over eight, many things may be taught him. Urinalysis certainly. Diet often. Self-administration of insulin can usually be taught children over nine, and the full significance of his disease with its complications of coma and shock, etc., is understood.

When the mother's schooling has been satisfactorily completed the child is sent home. The average first admission should not exceed two weeks. The Nutrition Worker visits the mother each day for a few days to help out with all the little odds and ends the mother had not thought of asking before. The urine specimens are carefully watched and if all the specimens are clear the insulin is slowly reduced until one slightly positive specimen is obtained a day. This is very important because a child's tolerance invariably improves after he gets home and starts full physical activity and a severe insulin reaction is apt to destroy the mother's confidence in her ability to handle her child and it may be very difficult to get her full co-operation afterward. The child is seen in the clinic, once a week for a month, at which time 24 hour specimens are examined and any slight adjustments in insulin which are necessary are made. The child's weight is watched to make sure that he is gaining satisfactorily. At each visit he is quizzed about insulin; about shock, about what to do if he has a fever, etc. Then the child is discharged to be seen once in three months for a year, and finally, twice a year. The place for the diabetic child is at home and not in the hospital, and once he is master of his disease he is far better off away from a hospital.

This seems like a great deal of work and fuss. The results would seem to justify its necessity. Nine of our cases have never been re-admitted to hospital for any reason whatsoever since their first admission in coma. One child has never been in hospital since he first came in in coma in 1930 at the age of eighteen months. Two have been re-admitted for infectious disease (Measles and Mumps), one for circumsision and one for tonsillectomy. Only three have been re-admitted in actual coma since their first admission.

That, in brief, is an outline of the way in which the clinic operates. And now for a consideration of some of the clinical features which these children present.

To date, we have had twenty-five children pass through the clinic. The youngest was eighteen months, the oldest was twelve years. Of these, fifteen were boys, and ten were girls.

ETIOLOGY

The cause of Diabetes Mellitus is not known. In the adult obesity, gall-bladder disease and thyroid pathology play a part; none of these factors play a part in childhood diabetes, as far as we know. In children, the important factors are:

1. Heredity.
2. Acute infections.
3. Endocrine pathology.

Heredity is undoubtedly a factor of the very greatest importance. Eleven of our twenty-five children had a family history of diabetes. One child gave a history of diabetes in five members of his family. It should also be remembered that the longer a diabetic child lives, the more diabetes do we find in his family. In one week last year, diabetes was diagnosed for the first time in relatives of two of our diabetics. It is not sufficient to ask a diabetic child about diabetes in his family on his first admission alone—each year the family history should be re-checked from this point of view. In addition the whole family of a diabetic child should be examined periodically for glycosuria. Joslin reports 53% of 73 diabetic children who have lived more than ten years have hereditary diabetes. Diabetes occurred seven times more often in the relatives of a diabetic child than in the relatives of a non-diabetic. We are only at the beginning of a study of heredity in diabetes. Prior to 1922, few diabetics married, and still fewer had viable children. Sterility was the rule. Under insulin therapy, diabetics are getting married in ever-increasing numbers and raising families, and it is from a study of their progeny that a final determination of the role of heredity in diabetes will be made.

Infection: The role of acute infections in the etiology of diabetes is a difficult one to appraise. Every child has two or three acute infections each year and yet the incidence of diabetes in childhood is very, very small. It is so easy to blame diabetes on the acute disease preceding it. We know that an acute infection may aggravate diabetes in a person who has it, but does an acute infection actually precipitate the disease? In our own series, as a rule, the incidence of acute infections preceding the onset of diabetes was, if anything, less than in the general childhood population.

Analysis of blood sugars has shown that acute infection, as a whole, does not affect sugar tolerance in children, except in mumps. Mommser and Mayer have shown that repeated blood sugar curves in children with mumps showed a diabetic

curve in severe cases on the fifth or sixth days. This did not occur early or late in the disease or in mild cases. Only one of our diabetics had mumps preceding onset of his disease, and in his case it preceded the diagnosis of diabetes by two years. One other developed mumps three years after diabetes had been diagnosed and suffered a marked loss in tolerance, which has persisted ever since.

Endocrines: The role of the endocrines, particularly the thyroid and the pituitary in the etiology of diabetes, is far from clear. The association of acromegaly with diabetes (25%), the frequency of over-height in diabetic children (86% Joslin), the production of hyperglycaemia during stimulation of the pituitary — all these have been brought forward as evidence that other endocrines besides the pancreas are at fault in this disease.

The whole problem of the relationship of the pituitary to the pancreas is still in the process of solution.

Diagnosis is comparatively simple. The presence of glycosuria with a fasting blood sugar of 140 mgms. or more, is definite evidence of Diabetes. The onset of the disease in childhood is very similar to its onset in adults—a history of excessive thirst, frequency of urination, progressive loss of weight,—these symptoms make a urine analysis for sugar imperative. One of our cases was treated as a nutritional problem for several weeks before sugar was looked for and discovered in the urine. Most of our cases did not consult a doctor until coma had supervened.

Diabetic Coma: The management of diabetic coma forms an important part of childhood diabetes, since most children are still first diagnosed in coma, and coma is still the most important cause of death in diabetic children. In our series of twenty-five children, coma has been encountered twenty-seven times. It is necessary to define just what is meant by coma. In some clinics a CO_2-combining power of twenty or less is regarded as coma—a purely arbitrary figure chosen because in the pre-insulin era the mortality in such cases was exactly 100%. In our clinic, because we had no access to CO_2-readings, a clinical standard was set up and any diabetic child showing drowsiness, hyperpnoea and ketonuria was regarded as a case of coma. As a matter of fact, only about one-third of the coma cases were actually completely unconscious.

The clinical picture of diabetic coma is too well known to all of you to warrant detailed description here. The success in treatment of this condition depends upon its being handled as a medical emergency. Absolute co-operation between the doctor, the house physician and the nursing staff is essential to a successful termination of the case.

I say house physician, and not house physicians advisedly. I believe that it is the duty of the interne who first sees the case to look after the case himself until the patient is out of coma. The man who starts the treatment is the one best able to judge whether the patient is responding satisfactorily to treatment or not. In a condition where insulin dosage and the administration of fluids must be varied from hour to hour, it is disastrous to have the case handled by two or three different internes.

As soon as the patient is brought into the hospital and the diagnosis made, insulin is administered at once. The dosage depends entirely on the patient's condition and varies anywhere from 20 to 40 units subcutaneously. We have given insulin intravenously on one or two occasions, but have discontinued this method of administration. The patient's bladder is emptied either voluntarily or by catheterization. It is important to start working with the bladder empty for a reason which will become obvious in a few minutes. The patient has a gastric lavage with 2% sod. bicarb. This is a procedure which we now carry out routinely in every case on administration, unless the degree of acidosis is very mild. We feel that if gastric lavage is not done early it will be done late. Lavage removes any food present in the stomach and very often enables the patient to take fluids by mouth thereafter, without vomiting. There is, however, a much more important angle to it. One of the commonest symptoms complained of by a patient in severe diabetic coma, is abdominal pain, often of an excruciating nature. The cause of this pain has never been definitely determined, but there are some people who think it is due to acute dilatation of the stomach—a part of a general intestinal ileus occurring in a coma. It is very possible that the occasional case of death in diabetic coma is due to acute dilatation of the stomach. At any rate the pain is very often promptly relieved by lavage, vomiting stops, and fluids can be given by mouth. If lavage is delayed, the patient may take fluids for a while, then vomiting sets in, treatment has to be interrupted for a gastric lavage, and valuable time has been lost.

An enema is then given, also routinely. This clears out the lower bowel and enables us to give a stimulant in the form of three to four ounces of warm black coffee as a retention enema. We make no effort to give fluids by bowel.

Blood pressure is taken and checked periodically. This is of a great deal of importance and a blood pressure, within normal limits, is a very valuable indication that the patient is coming along satisfactorily. Conversely, a falling blood pressure is a sign of very serious significance, since with a falling blood pressure, kidney function is impaired and one of the main avenues of excretion of ketone bodies is closed.

The above-mentioned procedures take up about twenty minutes; they can be done without fuss and without hurry and rarely have to be repeated.

From then on treatment is a matter of:

1. Supplying fluids to combat dehydration.
2. Giving insulin to overcome the acidosis.

3. Supplying warmth and vascular stimulation.

Fluids are given by mouth whenever possible and, very often it is possible. Bovril, salted broth, 5% glucose drinks, ginger ale, water, are all given in sips at first, and gradually increased. The nurse must be wide awake enough to see that the patient is offered fluids every 10 to 15 minutes. Glucose is not forced or given in any definite quantity. My feeling is, that there is already an abundance of sugar in the blood and it requires only sufficient insulin and an improved circulation to render it available. No attempt is made to cover insulin with glucose, so long as there is marked sugar in the urine. When the sugar begins to disappear in the urine, fluid carbohydrate, in the form of orange juice, glucose drinks, or ginger ale, are given to the amount of 100 gms. in twenty-four hours. We *do* measure the amount of fluid taken, to be sure that sufficient fluids are given. In addition to giving fluids by mouth, an intravenous of normal saline is often given at the outset, in a case of moderate severity. The solution must be allowed to run in very slowly. Occasionally, we have used the continuous intravenous method, allowing solution to run in very slowly over the course of about twelve hours.

Insulin is, of course, an extremely important factor in treatment. We can give no rules as to the quantity of insulin given or the frequency with which it should be given. Our cases take, on an average, 100 to 150 units in the first twenty-four hours. We have had cases come out of coma with 50 units, and we have used 200 units. The dosage is determined as the result of urinalysis and clinical observation. As I pointed out earlier, treatment starts with an empty bladder. Thereafter, every dose of insulin is preceded by a urinalysis for sugar, diacetic acid and acetone. As long as sugar is present in the urine, it is safe to give insulin. If sugar disappears from the urine before the patient is out of coma, then glucose must be given by every available route until sugar reappears in the urine.

Insulin is given every hour or every two hours until the urine tests show that the sugar is decreasing. Then, we follow Joslin's scheme of giving 20 units for a red test, 15 units for a yellow test, 10 units for a green test, and no insulin, of course, for a negative test. Insulin at this time is given every four hours.

There may be some here, who will disagree with some of the details of treatment of coma in children, as carried out in this clinic. It is only necessary for me to say that since the clinic was organized in 1930, there has been not one single death in a diabetic in the Children's Hospital.

Complications of Coma.

I have had some interesting experiences in our coma cases, but I propose to refer to only two of these.

ABDOMINAL SYMPTOMS OF DIABETIC COMA

One of the most interesting, as well as one of the most puzzling conditions met with in diabetic acidosis, is the so-called diabetic abdomen. This condition has been encountered on several occasions, and is far more common than is generally realized.

CASE 1. A girl of eleven who had had Diabetes for two years, was perfectly well on Sunday. On Monday, she felt poorly and vomited her breakfast; she stayed in bed and felt somewhat better. Tuesday morning she vomited again, had some difficulty in breathing, and began to experience severe right-sided abdominal pain. She was admitted to the Children's Hospital. Examined by the interne, she revealed a picture of moderate acidosis, a rigid right abdomen, and marked tenderness over the whole right side of the abdomen. The interne's diagnosis was Diabetic acidosis, due to either acute appendicitis or lobar pneumonia involving the right lung base. Leucocyte count was 30,500. Temperature was 102 degrees. Urine was loaded with sugar, diacetic acid and acetone. Gastric lavage was done, and 40 units of insulin given, along with glucose and intravenous fluids. When she was seen four hours later, she was bright, had no pain, and the abdomen was completely relaxed.

CASE 2. A girl of 13, a known diabetic, was brought into the Children's Hospital, with entrance complaints of drowsiness, severe right-sided abdominal pain, and vomiting. Examination showed a girl in moderate acidosis, drowsy but easily roused. The right side of the abdomen was rigid and very tender on pressure. The abdomen was distended and it was thought that the apex beat of the heart was displaced upwards and to the left. The leucocyte count was 30,000. Temperature 101 degrees. Pulse 140. Urine loaded with sugar, diacetic acid and acetone. Gastric lavage was done, insulin and fluids given, and she improved rapidly. Twelve hours later, she was fully conscious, free of pain and had a leucocyte count of 9,800.

These two cases serve as examples. Usually there is no difficulty in diagnosis, since the acidosis overshadows all other symptoms. Sometimes, however, the abdominal symptoms form the prominent part of the picture and diagnosis may be extremely difficult. The leucocyte count is more often a hindrance than a help, since our experience is that it is always elevated in diabetic acidosis sometimes as high as 50,000 or 60,000. When one realizes that it is perfectly possible for a diabetic to have an attack of acute appendicitis and develop acidosis as a result, the importance of caution in the making of a diagnosis becomes evident, operation is not to be considered lightly in a patient already in acidosis. I believe it is safe to treat these patients for four hours as simple cases of acidosis, as in my experience, the abdominal symptoms have usually cleared within four hours of the commencement of treatment. The diabetic who has acute appendicitis usually presents pain as the first symptom,

rather than vomiting, and the tenderness is usually definitely localized. Rectal examination may be very valuable in differentiating. If the abdominal symptoms persist after four hours of intensive treatment with insulin and fluids, laparotomy should be done. I feel there would be nothing lost by the delay since the patient, in any case, would be a much better operative risk with the acidosis controlled.

The cause of the abdominal symptoms in diabetic acidosis has never been satisfactorily explained. Three theories have been put forward:

1. That it is due to an inflammatory process in the pancreas.
2. That it is an acute diabetic neuritis.
3. That it is due to acute dilatation of the stomach.

I am inclined to favor the last of these theories. A generalized intestinal ileus of greater or less degree seems to occur in every case of coma that I have seen. Enemas are necessary to empty the bowel and abdominal distension is frequent. There is frequently prompt relief following a gastric lavage and the quantity of brownish fluid present in the stomach in these cases, is often surprising.

Anuria.

Anuria is a dreaded complication occasionally met with during the course of treatment of a case of diabetic coma. We have seen it on two occasions.

CASE 1. A girl of 12 was admitted in coma on November 25, 1932. She presented the typical picture of diabetic coma with Kussmaul respirations, soft eyeballs, etc. Sugar, diacetic acid and acetone were present in the urine in large amounts, and micro-urinalysis also showed many granular casts (a not uncommon finding in diabetic coma). Leucocyte count was 61,000. The girl's condition was very poor. An intravenous of 5% Glucose in saline was given and insulin was given intravenously, at 11 a.m. Insulin was repeated at hourly intervals and then at two-hourly intervals, and she seemed to improve during the afternoon and even took fluids by mouth. In the early evening, the interne (who, by the way, had been on duty on the case from the time of admission), noticed a change in the patient's condition. The girl was in deep coma but her breathing was shallow and she could no longer be roused to take fluids. In addition, he noticed that he was obtaining very little urine by catheterization and, in fact, none from 7 p.m. on. I saw her at 10 p.m. Blood sugar and blood urea were taken for scientific purposes and a continuous intravenous of 10% glucose started. Shortly thereafter, urine began to appear again, and by morning, she was conscious and voiding normally. The laboratory, the next morning, reported a blood sugar of 177 mgms. and a blood urea of 164 mgms. per 100 ccs.

CASE 2. A child of four, admitted in diabetic coma on February 15, 1934, at 9 p.m. Intensive treatment was started, and by next morning she was conscious and taking fluids readily by mouth. At 6 p.m. she was bright, talking to her mother, and seemed quite well. The next day she lapsed into coma, although, the clinical picture was not that of diabetic coma. Her respirations were shallow, pupils contracted, and she could not be roused. Catheterization produced about two drams of urine containing no sugar, but containing acetone and diacetic acid. Blood urea was taken. An intravenous of 50 ccs. of 25% Glucose was given. Duiresis followed soon after, and the patient made a complete recovery. The blood urea report was 100 mgms. per 100 ccs.

The exact cause of anuria in diabetic coma has not been satisfactorily explained. The following factors have been suggested:

1. Circulatory disturbance in the kidney, accompanying a decrease of blood pressure and cardiac failure.
2. Dehydration of blood plasma as the result of lack of intake of fluid, polyuria and vomiting.
3. Acute pathologic change in the renal parenchyma, as a result of an excessive excretion of ketone acids in the early stage of coma.

Success in the treatment of this dangerous complication depends on early diagnosis and prompt treatment with hypertonic solutions of either saline or glucose—both methods having been used with success.

Blood Sugars.

You may have noticed that no reference was made to the taking of an emergency blood sugar on admission. We have tried to treat our cases of coma without the assistance of blood sugars. Originally, this was done for the purpose of teaching internes to treat these cases as they might to treat them in general practice away from a hospital. We found that we could usually get along very well without a blood sugar report. After all, a blood sugar taken on a patient coming in in coma can only do two things: it can make the diagnosis, and it can satisfy one's curiosity. In over 95% of the cases, the diagnosis can be made readily on history, physical examination, and urinalysis and usually is made before ever a blood sugar is taken. From the patient's point of view, the satisfying of the doctor's curiosity is an expensive procedure. It makes very little difference whether the blood sugar is 300 mgms. or 500 mgms., and, in any case, a laboratory report showing that the blood sugar is 300 mgms. only means that it *was* 300 mgms. at the moment the blood was drawn. An hour may have elapsed in the interval, insulin given, glucose and fluids given, gastric lavage and enema done, so that the blood sugar report is of very little value. Blood sugars are sometimes taken a little later on in treatment when the urine is clearing and there is some doubt as to whether it is safe to give more insulin or not. Then the blood sugar

is of more definite value and may be justified. To take the place of the blood sugar, the urine is used as a guide. The treatment of a case of diabetic coma is started with an empty bladder. Every dose of insulin is preceded by a urinalysis for sugar diacetic acid, and acetone. So long as sugar is present in the urine, one can safely assume that the blood sugar is over 160 mgms. and insulin can be given with safety. As the urine tests show less and less sugar, insulin is given less often, and in smaller doses.

The management of a case of Diabetes calls for a consideration of three factors:

1. Diet.
2. Insulin.
3. Exercise.

Diet: A great deal has been written about diet and diabetes, and about a dozen different types of diet have been used in the treatment of Diabetes. We shall concern ourselves with only four.

1. The high fat diet of Newbergh and Marsh.
2. The high carbohydrate: low calorie diet, as used by Sansum, Geyelin, Rabinowitch.
3. The free diet.
4. The normal type of diet.

The High Fat Diet was particularly popular in the pre-insulin era, and served a definite and valuable purpose. It is, however, particularly unsuited to children. It is unpalatable. It is not the type of diet the other members of the family are having, so that the diabetic child is continually being reminded of his disease. The large percentage of fat and the correspondingly low percentage of carbohydrate, makes the child a candidate for acidosis everytime he develops an acute infection. It is a diet which leads a child into temptation and disorganizes diabetic management. Normal growth is the exception rather than the rule.

The High Carbohydrate—Low fat diet, we feel, is just as wrong as the high fat diet. It is not a normal type of diet and the restriction of fat deprives the patient of some foods which he likes. Unless the fat is kept at a very low level, excessive gain in weight will occur, and we have found it impossible to control glycosuria in this diet.

The Free Diet is mentioned, only to be condemned. A diabetic on a free diet is an uncontrolled diabetic. He dispenses first with his scales, then with his urine tests, usually with his doctor, finally with his life. The result is recurrent acidosis and the development of all the dreaded complications of the disease. Training and discipline are important features of management of a diabetic child. With a free diet there is no place for either.

The Normal Type of Diet is the diet we favor. Its advantages can be summarized:

1. The diabetic's diet is the same as the diet of the rest of the family, and therefore it is less expensive.
2. It is a palatable diet.
3. The child can have every food he likes included in the diet.
4. It allows for normal growth and full activity.
5. It furnishes a measure of protection against acidosis.

Our aim has been to give the child a diet which will be satisfactory to him and to us, which will allow him to attend school full time and take part in all the activities in which he is interested; which will allow him to grow normally in height and weight, so that he cannot be picked out of a group of his playmates and labelled a diabetic, and which will not impose upon his mother the added burden of preparing two separate menus for her family. The normal type of diet has filled all these expectations, and we have had no desire to change it. Each diet must be adjusted to each individual child and there are no text-book formulae which will tell you how much carbohydrate, protein, and fat a child requires. The children in this series are on diets ranging from 100-165 gms. Carbohydrate, 60-75 of Protein, 70-90 of Fat.

Insulin: Of the use of insulin I need say only that it is used in sufficient quantity to enable a child to take the diet he requires for normal growth and full activity, and not excrete more than 25 gms. of sugar in the urine per day. Every childhood diabetic requires insulin. If insulin is withheld either because of the parent's obstinacy or the doctor's desire to get along without it, the child must necessarily be kept on a restricted diet which will interfere with growth and nutrition, or if he is kept on a full diet, his disease will progress to a fatal termination. The benefits of insulin have not been fully realized by the medical profession, and there still are men who use insulin only to rescue patients out of coma, and then pride themselves on getting along without it. Insulin is available and it should be used to full advantage. Some of our children use 3-4 units a day, others use as much as 60-70 units a day. Most of these take two injections a day, some take three, and one takes four. Three children are now taking Protamin-Zinc-Insulin instead of the old insulin. These cases have not been studied long enough to be included in this report.

Exercise is very important in the management of the chidhood diabetic. Exercise tends to lower the blood sugar of a well-treated diabetic. Children in Diabetic summer camps get along on less insulin than they require during the winter, when exercise is necessarily restricted. We encourage these children to take part in all activities: Hockey, skating, football, and so on.

CONCLUSION

Our purpose in establishing the clinic was not to collect a great mass of statistics or to attempt to revolutionize the treatment of Diabetes. We hoped, by co-ordinated effort, to study the disease with particular emphasis on the management of the child outside the hospital. We felt

that it was possible for these children to lead normal, happy lives; that home management could be simplified by proper education and follow-up; that the disasters of diabetic coma and continuous hospitilization could be avoided by properly organized teaching, and the creating of a sense of responsibility in the mind of the child and its parents. Finally, we hoped in the course of time, to be able to give the lie to those who felt that a diabetic child was better off dead. There are many in the medical profession today who look upon a diabetic child with mingled feelings of pity and pessimism, for which, I believe, there is no justification. Diabetes of childhood has gone through three eras—the era of complete hopelessness, which prevailed prior to 1922, when a diagnosis was a seal of doom and when a man like Van Noorden was able to say:

> "If a true diabetic disposition develops in a child, it progresses relentlessly, however slight it may be at first, and even when the treatment is the best in the world. Infantile diabetes knows no mercy; one can, at most, only defer the end for a little time."
>
> —*Van Noorden*, 1905.

Then the opening of the insulin era, when it became possible for a diabetic child to live and be kept alive, but on diets so inadequate, and on doses of insulin so small, that life seemed hardly worth while. And, finally, the present era, when, with the aid of sufficient insulin, normal diets and proper management of the disease, the diabetic child found it possible not only to live, but to enjoy life in a measure often denied to his more fortunate non-diabetic playmate.

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*Tuberculosis in Northern Manitoba

By

E. W. MONTGOMERY, M.A., M.D.,
C.M., LL.D. (Man.),
Professor Emeritus in Medicine,
University of Manitoba

The care of tubercular patients is the most important problem in public health administration, because the death rate from tuberculosis is higher than from all other infectious diseases combined. Patients who are ill with tuberculosis are ill a long time, and if they recover the recovery is often only partial, a condition of chronic invalidism and incapacity being the final result of years spent in taking the cure. The cost of tuberculosis clinics, sanatorium beds, public health nursing, etc.—the shock troops in the war against tuberculosis—is a staggering amount. In this Province of 731,000 people this front line warfare is carried on at a cost of $500,000 a year.

This amount of five hundred thousand dollars a year is a very conservative estimate of the actual amount expended in the care of tuberculosis people in this Province. For example, last year the Ninette Sanatorium and the Central Tuberculosis Clinic combined spent about two hundred and thirty thousand dollars; the St. Vital Sanatorium about the same amount, perhaps a little less. In addition to this, there was a very considerable number of tuberculosis patients looked after in the King Edward Hospital, Winnipeg. Besides this, in every hospital in the Province there are cases of tuberculosis being maintained at public expense, some of them temporarily while waiting for admission to the Sanatorium and others with various forms of tuberculosis which may be treated more advantageously in a general hospital than in a sanatorium. Then the Public Health Nursing service devotes a considerable part of its activities to the care of tubercular patients outside of institutions. From these facts it is very obvious that the cost of looking after the tubercular in Manitoba is considerably above the figure mentioned.

It is interesting further to note that the per capita ward cost of patients in public wards throughout the Province last year was $2.30 per day, of which 60c per day was spent for the care of the tuberculous, while in Unorganized Territory, which is situated almost entirely in the northern part of the Province, the cost for tuberculosis amounted to $1.17 per day, just about double what the actual cost was in the southern part of the Province, indicating the intense segregation of the tuberculous in northern Manitoba; in other words, the percentage of cost for looking after tuberculous patients in the organized portion of Manitoba was 27% of the whole, while the cost in Northern Manitoba was 42% of the total cost.

In addition to this the wives and families of these patients are usually supported from public funds, and who is prepared to estimate how much the community loses through the impaired activities of discharged tubercular patients? The taxpayer is usually called upon to foot almost the entire bill. During a three year period in a large local sanatorium less than 1% of the patients paid cost for services rendered, and less than 3% made any direct financial contribution to the institution.

The people of this province have been educated to the point where they have given generously to carry on a campaign having for its objective the complete control of tuberculosis within a short time, but this objective is not being reached as expeditiously as was hoped. We find that in the race for a low death rate Manitoba and British Columbia are being outdistanced by the other western provinces—even by Ontario—while seven States immediately adjoining us and south of the International Boundary, have a yearly death rate 43% less than ours.

Tuberculosis Mortality Rates per 100,000
For the Year 1934

Prince Edward Island	104
Nova Scotia	88
New Brunswick	66

* Read at the Annual Meeting of the Manitoba Medical Association at Winnipeg, May, 1936.

Quebec	88
Ontario	37
MANITOBA	**53**
Saskatchewan	30
Alberta	40
British Columbia	78

A study of mortality rates as affected by racial groups points very clearly to one of the causes for our poor showing in the contest. We have in Manitoba a larger Indian population than any other western province, except British Columbia. Roughly 2% of our total estimated population of 731,000 is composed of Indians, and this 2% contributed 28% of all the deaths from tuberculosis in Manitoba in 1934: To be precise, there were 113 deaths among 15,000 Indians—a rate of 753 per 100,000—while among 716,000 others of the population there were 282 deaths, a rate of 39 per 100,000!

The geography, and especially the climate of any country, is often a very important factor in determining the living conditions of the inhabitants, so in order to get a clear understanding of public health problems in Manitoba it is necessary to review briefly the geography and climatic conditions of the Province.

Manitoba is the only one of the three prairie provinces that has direct access to salt water. Churchill, at the mouth of the Churchill River, has an excellent harbor opening into Hudson Bay, but navigation is closed by ice for eight or nine months out of the year, and although Churchill is on the same parallel of latitude as the northern part of Scotand, 58° north in one case means nine months winter, and in the other, harbors open twelve months in the year.

The entire province of Manitoba is drained by rivers which flow into Hudson Bay, and since there is a gradual slope of land northward, and no intervening mountain range, cold north and west winds prevail. The rainfall, while not abundant, is usually sufficient for the growth of hard grains and the soil in the south and western prairie sections is excellent. All of north eastern Manitoba, probably five-sixths of the whole province, is part of a gigantic peneplain, which extends from Labrador to the Rocky Mountains. The country rock is almost entirely igneous: granite and gneiss, etc. In consequence the soil is acid, the forests are predominantly coniferous, sparse, and usually stunted, especially towards the northern sea.

The landscape of northern Manitoba owes its characteristic features to intense and repeated glacial erosion. Thousands upon thousands of lakes are scattered broadcast among the granite hills and primitive rocky ledges which slope down to Hudson Bay. Many rivers, all flowing north and east, carry on the work of wearing down the old Laurentian Highland and transporting its sediment into the shallow southern end of the Great Bay of the North. Scattered throughout this vast region of lakes, rivers and dark forests of pine and spruce is a meagre population of not more than twenty thousand souls. Half of these are Indians, with the balance largely composed of miners, prospectors, white trappers, and traders, and a small number of employees of the Hudson Bay Company. The Indians are Wood Crees, an offshoot of the Great Ojibway stock. Dr. J. C. B. Grant, now Professor of Anatomy in the University of Toronto, a few years ago spent a summer among these Indians, measuring and studying their anatomical peculiarities, with a view to determining their affinities, either to the Indians of the North Pacific Coast or to the Plains Indians to the south. I believe it was Doctor Grant's opinion that the Wood Crees had come from the south, rather than from the west, and probably they were driven down the Arctic slope by the more vigorous tribes of the Great Central Plain. The Crees did not trek into this wilderness of lakes, sparse vegetation and turbulent rivers flowing through granite gorges, because they were drawn by the urge of a pioneer to enter a promised land; rather they fled north to escape the arrows and spears of the mounted Mandans and Sioux. The Crees live in winter in log shacks, the logs chinked with moss. The roof is also of chinked logs covered with earth. There may be a window and an ill fitting door. There never is a floor. Into this wretched hovel a hunter and his family, composed of children, nondescript adults, and dogs, huddle together through the long winter nights. The head of the family is out following his line of traps, sometimes not returning to his hut for a week. The physical strain these unfortunates must endure can only be realized by one who has personal knowledge of the facts.

The treatment of tuberculosis is not satisfactory because its etiology is still obscure; its immediate cause—infection by the bacillus tuberculosis—is of course a matter of common knowledge, but the tuberculosis bacilli is only the seed which requires a fertile soil in which to germinate, and what is a fertile soil?

In many areas, both urban and rural, in North America, there are consumptive centres, where both mortality and morbidity rates are inordinately high. What are the common factors in living conditions in these infected areas? Evidently bad hygienic surroundings, crowding, poor ventilation, whether in isolated Indian shacks on the shores of Hudson Bay or in a crowded tenement in New York. Improper and insufficient food. This applies particularly to the Indian population of the Northwest. Fish is their staple food, mainly whitefish in summer. The fish is netted, cleaned and dried in the sun, then ground up into a kind of pemmican, and kept in reserve for times of scarcity. Many Indian families live almost exclusively on fish, but if the fur crop is profitable a litte flour and tea is obtained from white traders. No vegetable food is used, although in the month of August, 1931, the writer found fourteen varieties of wild fruit ripening and in great abundance at latitude 57 north! It is a curious, perhaps an illuminating fact, that the Esquimaux who live north of 60, and who live on an exclusive diet of sea fish and seal meat, are practically exempt from tuber-

culosis. May we not logically assume that a diet of fresh water fish lacks some essential element which is found in the flesh of fish caught in salt water? After living for a prolonged period—a month or more—on fish, the Indians become greatly emaciated and most of them suffer from diarrhoea; then certainly the soil is well prepared to produce an abundant harvest of consumptive patients.

The Indians of the North-west are handicapped against tuberculosis because of:

(*a*) Poor heredity,
(*b*) Inhospitable climate,
(*c*) Bad hygiene,
(*d*) Improper food.

What can be done about it?

The stock is not being improved by intermarriage with the class of whites who condescend to such alliances. Neither does marriage with the halfbreed population serve to introduce a more resistant strain, as shown by the fact that the tuberculosis death rates are even higher among halfbreeds than among Indians.

There is not much that can be done to remove the climatic or environmental handicaps of the northern Indian. For years the Federal Government of Canada has maintained a considerable number of boarding and industrial schools where Indian boys and girls receive an elementary academic education. They are also taught domestic science, and are given besides a course in farming and certain trades. If these boys and girls would remain down where the land is arable and the seasons suited to the growth of cereals and live stock, much good might be accomplished. "But" and "if" are the most significant words in the English tongue. An Indian youth has a background of a hundred generations of hunting, fishing, and loafing in the sunshine, and who could expect to uproot traditions of a race of such great antiquity in a few short months of intensive training? The writer has spent several summers in the north visiting remote trading posts, travelling by canoe, following the maze of lakes and rivers that extend from Lake Winnipeg to the Bay. Game is scarce, forest fires and wasteful methods of hunting and trapping have almost exterminated the beaver, and with the beaver has gone at the same rate the less valuable fur-bearers, so the Indians' occupation has vanished with the beaver. He, perforce, must live anyhow or somehow, and now he camps in the neighborhood of the white man's trading posts, mining camps or fishing stations. He lived precariously even at the best; now there is no best, nor hope of it, for the aborigine. There seems to be but one ray of hope; fur farms are now producing a considerable fraction of the world's supply of fur. Why should Indians not be taught how to farm foxes, beaver, mink and muskrat, instead of striving vainly to make of them bankrupt dirt farmers?

Volumes might be written about the history of tuberculosis on this continent. Did the Paleface infect his Red brother with the deadly bacillus of tuberculosis as well as the potent treponoma pallida, or were the North American Indians subject to tuberculosis previous to the coming of the whiteman? In 1633, only 25 years after Champlain founded the Colony of Quebec, Le Jeune, a Jesuit missionary, found that nearly all the young Indians had scrofula, and less than forty years later Jessel found that among the Indians of New England consumption was extremely common. Nevertheless it is quite possible that tuberculosis was introduced from Europe after 1492, and spread like wild fire among the non-immune natives of the western world. According to Dr. George Ferguson of Saskatchewan, who has studied the history of tuberculosis as affecting Indians of the western prairies, there is little or no evidence of the existence of the dread disease among the Indians of Satskatchewan prior to 1870, but by 1884 the infection had become an epidemic of the most lethal character. Since then the virus has become less potent, or the soil less fertile, until now the mortality rate in Saskatchewan in considerably lower than it is among the Indians of Northern Manitoba. The Indians of Saskatchewan are of a more vigorous stock than those who inhabit the wooded area to the north-east, and would in consequence develop more rapid resistance to infection.

Reference has already been made to certain Boarding or Industrial Schools which our Federal Government maintains for the education of Treaty Indians. Doctor Meltzer, of the staff of Ninette Sanatorium, has recently completed a survey of health conditions in six of these "Indian" Schools. Five hundred and twenty-seven children between the ages of two and twenty were Tuberculin tested, had chest plates made, and were subjected to physical examination. A second group of three hundred and forty-six white children attending school, and known to have contact with active cases of pulmonary tuberculosis was examined, and finally a group of three hundred and ninety-one unselected white children were subjected to the same series of tests, and a comparison made of the findings in the three groups:

1st—527 Indians at school
2nd—346 white children, known contacts
3rd—391 white children, unselected

Many interesting points of comparison were demonstrated by a tabulation of the results obtained, but just a few of such contrasts may be referred to here.

(*a*) **Tuberculin Reaction:**

Group 1—440 out of 527 Indians reacted 84%
Group 2—189 out of 341 whites reacted 56%
Group 3— 65 out of 391 whites (unselected) reacted ..16%

(*b*) **Clinical x-ray evidence of Tuberculosis:**

Group 1—Some evidence of Tuberculosis infection 72%
Group 2—Some evidence of Tuberculosis infection ...39%
Group 3—Some evidence of Tuberculosis infection 11%

Among other suggestive facts disclosed by Doctor Meltzer's survey may be mentioned the finding that in schools on the reservations—where the pupils are in more or less intimate

contact with their parents—direct evidence of tubercular infection was at least twice as frequent as it was in children at schools where schools were centrally located and a long distance from Reservations, hence few opportunities for contact and infection. This fact also indicates that the Indian children of non-resistant stock react very favorably to good food, warm clothing and the excellent general care which they receive in Government Schools.

The crux of the situation seems to be this: The Province is doing a great deal in the way of providing hospital care and public health and welfare services for its white population, while a steady stream of infection from the North filters through the fringe of metis or half-breeds which lies between white and Indian areas, and as long as no attempt is made to control this infection at its source, little improvement can be expected. The Hudson Bay Company employees, traders, trappers, and explorers of many European peoples have for several centuries intermarried with the Indian women, and have left a numerous progeny of half-breed children, many of whom, when they reach adult life, become Indians in all but color, and live by trapping and fishing. Some of these hybrids, however, settle down as farmers and traders, and are gradually assimilated into the Caucasian races. Along the great rivers of the North—the Saskatchewan, the Red and the Assiniboine—settlements of natives (whites and half-breeds) have existed for many years. These half-breed communities have been a sort of genetic halfway house between the two races—white and red—and through this medium the white man and the red have interchanged infections as well as other commodities, desirable and otherwise. As a consequence these half-breed settlements are probably more heavily infected with tuberculosis than even the Indians of the North.

The situation with regard to tuberculosis is almost as bad in certain sections of the half breed population as it is amongst the Indians. For example, a survey was made in 1932, of a small municipality with a population of 520. One hundred and eighty-seven applicants were examined, of which number 26 were found to have active infection. Of these 26 patients, ten were admitted to a sanatorium at once, and within the next few months six more, making sixteen cases of active disease that require prolonged institutional care. This means an actual expenditure of over $10,000.00 a year.

There are doubtless many other sections of northern Manitoba where almost parallel conditions exist; and it is evident, if the Province undertook to hospitalize all of this class of patients, who need institutional care, the cost would be far beyond the resources of the taxpayers at present. There must be some alternative way of dealing with this particular angle of the problem. Something in the way of a Public Health Nursing Service, such as has been established in Western New York State under the Millbank Memorial Fund. The writer visited Cattaraugus County in 1928, and believes that the type of service established there in a rural community might, with modification, be adapted to our own Province.

The logical way to solve the problem of high death rates from tuberculosis in this province is evident. Stop the spread of infection from the North. But how? There were 113 deaths (from tuberculosis) among 15,000 Indians in 1934. 113 deaths per annum in a white population would mean, by New Haven standards, at least 400 people sick with tuberculosis. In an Indian population it would mean more than 400 ill and requiring care. Frankly speaking no government—however altruistic in sentiment it might be—could spend the taxpayers' money at the rate of $800 per day in looking after tuberculous Indians, when all Indians, sick and well, number only about 2% of our total population. So a bed for every Indian sick with tuberculosis is a fantasy, a will-o-the-wisp, and not within the range of possibility.

Must we then admit defeat? Must we go on and on trying to cure our tuberculous whites and watching the deadly flood of infection pouring down from the North year after year? The day that Columbus touched American soil saw the Doomsday Book of the American Indian opened to record his fate. Ravaged by war, driven into the waste places of the desert and the Arctic barrens, decimated by pestilence, and harrowed by unspeakable poverty, is it not both a duty and a privilege of the conquering white man to make what amends he can for five centuries of injustice to the 100% American?

Tuberculosis is more than an infectious disease. It is a challenge to civilization, the answer to which is a world at war. The fighting units include not only laboratories, clinics and sanatorium beds, but also huge provincial and municipal budgets to defray costs. The problems demanding solution are most intricate—involving factors of social and political responsibility, factors which touch the thoughts and the pocketbooks of every citizen of Canada.

The purpose of this paper is to demonstrate the unique situation which exists affecting the control of tuberculosis in Manitoba. The various factors which contribute to the unusual prevalence of the disease among our Indian population are reviewed. The channels through which the infection filters into the white population are outlined, and some—unfortunately a very few—suggestions are made as to how conditions might be improved. The writer feels that present methods of control of tuberculosis among the whites are, for various reasons, not feasible nor adequate when applied to the northern Indians, and he is looking for more light on a problem, the answer to which up to the present moment is clouded with uncertainty.

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